THE

JOURNAL

OF

Nervous and Mental Disease

Vol. IV. APRIL, 1877. No 2.

Original Articles, Selections and Translations.

ART. I.—PATHOLOGY AND TREATMENT OF NEURALGIA.

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(Read before the Society of Physicians and Surgeons, Chicago, March 12*th,* 1877.)

NEURALGIA is one of the most common forms of disorder to be met with in the experience of the practicing physician. It has been written upon most extensively at various periods in the course of the history of medicine, and never more profusely than in the past few years. But after all it cannot be said that it is well understood, more particularly as respects its pathology.

In the present paper it will be my purpose to consider this subject, with some care, and with the hope of placing it in a clearer light than it has been by most who have written on it up to the present time. In the endeavor to carry out this purpose, it will not be possible for me to enter into a history of the progress of knowledge on this subject.

Neuralgia, as all are aware, signifies nerve pain, and implies always, at least in my opinion, a material lesion of some part of the sensory nervous apparatus. But what is its true nature, and its true seat in the nervous system, and on what morbid changes does it depend? What is its real mechanism or make up, and the various steps in the morbid process? It seems to me, that we are now in a position to answer these questions in a somewhat definite manner. But before I begin to describe what I believe to be the true pathology of neuralgias, it is necessary that I should refer to certain points in the anatomy and physiology of the nervous system, as a basis for what is to follow.

The sensory nervous apparatus in its simplest form consists of a conducting tract and a central apparatus of nerve cells, for receiving and appreciating the impressions conveyed to them, by the way of the conducting tracts. This simple statement covers the whole case. So far as present knowledge goes to show, nerve *fibres* perform no other function than that of conducting impressions. They cannot feel them, as it seems may be done by the cells of the sensory nuclei to which they lead. Sever a nerve trunk or fibre in any part of its course, from its appropriate centre, and no degree of irritation will cause the part peripheral to the cut to react to the impression, so as to give us what is known as a sensation. This is not a matter for surprise, for nerve fibres *per se* are not the seats of true nerve sensibility. Simple as this fact may appear to be, it is important for us to remember, and use, in our investigations into the pathology of neuralgias.

In its normal state, as respects its nutrition, a nerve fibre has its maximum of conductibility, or as M. Vulpian would say, *neurility*. It does not seem possible for a nerve fibre to be brought into such a structural state, by disease, as to augment its conductibility, beyond what is natural to it, in a condition of health. But if there are doubts to be entertained, in respect to this point, there can be none, as to whether the conductibility of a nerve fibre may be diminished or even destroyed by disease. According to my view, this must in some degree, be always the result of morbid structural change in a nerve fibre. Any departure from its normal structural state, in which state it is most perfect as a conductor of impressions, in all probability, impairs this function. If this is a fact, as I believe it to be, it has an important bearing on the pathology of neuralgia, as I hope soon to show.

Let us now for a moment give attention to the central sensory apparatus of nerve cells. This is the true seat of nervous sensibility. Any impression, no matter how or under what circumstances produced on a nerve fibre, unless it shall reach this central mechanism of nerve cells, does not give rise to what is called a sensation. The capacity to recognize, or appreciate sense impressions, is found alone, at least in its perfection, in nerve cells. Certain it is, it does not exist in nerve fibres. No form or degree of disease can confer on nerve fibres this capacity, of which they are naturally devoid. If there is doubt as to whether nerve fibres can have their conductibility increased in any way above the normal level, there can be none, as to whether sensory nerve cells may have their sensibility rendered more acute than natural, or increased to an unhealthy degree, under certain circumstances. Neither can there be any doubt as to whether their sensory capacities may not be diminished or blunted by various means, healthy as well as morbid.

If the foregoing remarks are true, a decrease or loss as compared with the normal degree of sensibility in a part, may depend on *first*, a loss in conductibility of the related nerve fibres, without any involvement necessarily of the receptive capacity of the related nerve centre, or it may depend on a change in the condition of the nerve centre itself, without any unhealthiness of the nerve fibre as to its structure and conductibility. But an increase in the sensibility of a part does not find its explanation in the condition of the nerve fibres, but rather on a change in the condition of the sensory nerve cells themselves. The apparent increase in sensibility does not depend on a condition of the *conducting* tract, which might be supposed thus to magnify as it were the sense impression, as it passes along toward the sense centre, so as to exaggerate there, the effect that would otherwise have been produced under normal conditions, but rather on a change in the cells of the centre which heightens their receptive capacity and thus renders them more responsive, more easily disturbed, more unstable. In case then of any augmentation of sensibility above what is normal, we are led to fix attention on the centres rather than on the sensory nerves themselves. And this as I hope to show, is what must be done in a true pathology of neuralgia.

Excluding for the time being the special senses from our view, we will fix attention on what has been called *general sensibility.* It is found, among other parts, in the cutaneous surface. When we consider the general sensibility of the skin, we find it may be divided into different kinds. We may distinguish at least three kinds. 1. The sense of contact or of touch. 2. The sense of temperature. 3. And possibly the the sense of pain or of painful impressions.

These three forms of sensibility of the skin, and possibly others, may be variously increased or diminished, or even qualitatively changed without involving the same kind or degree of change in the other forms. Thus the sense to painful impressions may be impaired or even abolished, or otherwise changed, and yet the sense of touch or the sense of temperature remain intact. And the same may be said of the other forms of sensibility. It would be easy to give examples of these cases.

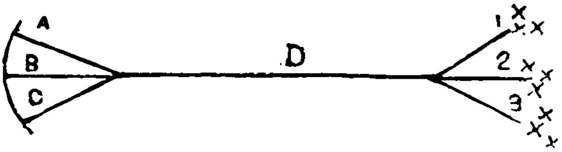
Now how can such phenomena be accounted for? How can we have the sense of pain either abolished or increased, without involving at the same time a parallel change in the other forms of sensibility which belong to the skin? Have we separate sensory nerve fibres to conduct the different kinds of impressions? If not, how can we account for the phenomena?

Dr. Brown-Sequard and others, hold, or have held, to the hypothesis, that these different kinds of sense impressions including all different kinds of sense impressions, have different kinds of nerve fibres to conduct them.

The sense of touch has one kind, and the sense of pain others, etc. If one set of these fibres should become diseased, then the form of sensibility to which they are subservient would be impaired or lost, while other forms of sensibility in the same part of the body might remain intact.

But there is another mode ef explaining the co-existence of different forms of sensibility in the skin, besides that of supposing they each have different kinds of nerve fibres. It is that the sensory nerve fibres of the skin are of one kind. These transmit indifferently and often simultaneously, different forms of sense impressions, such as those of tact or touch, and pain and temperature.

The physiological analysis of these different impressions, is made at the centre to which the nerve fibres lead. This notion may be illustrated, by a reference to the followng diagram:



Let A, B and C represent different kinds of cutaneous sensory impressions, produced on the peripheral end or apparatus of a nerve fibre. Let D represent the fibre along which the impressions are conducted. Next, let 1, 2 and 3 represent different groups of nerve cells at the central end of the fibres. In this case it is supposed that the fibre is made to connect, more or less directly, with all these groups of cells. The impressions of any or all forms, which are conducted along the fibre, are transmitted equally to all the centres or different cell groups. But though they all receive the three different kinds of impulses, yet each group is fitted to react to one kind of impression only, and none other. If the group of cells at No. 1 is fitted to react alone to impressions of contact, it will not recognize impressions of temperature, though they really reach it quite as forcibly perhaps as they do the cells of No. 2, which latter are fitted to react to them, but not to impressions of touch. In this way as can be readily seen, may we account for modifications of one form of sensibility without of necessity involving other forms closely related. If some modification should be made in the condition of the group of cells at No. 1, we might witness as a result, increase or decrease, or even a total loss of the sense of touch in the related parts, without the nerve fibre itself participating in any way. Happily we can illustrate this point in a very beautiful manner by a reference to the telephone of Mr. Elisha Gray, of our city. By it he is able, as we know, to communicate several dispatches—as many as eight—simultaneously over the same wire, and by a set of peculiar receivers to deliver them at the other end. This we know is done by communicating to each current a vibration which corresponds to that of a given musical note, and this vibration the current maintains faithfully to the point of delivery, where it is reproduced by a musical mechanism attuned to respond to it, and to it alone. And so in the transmission and central analysis of sense impressions. These latter phenomena do not depend on the conducting fibre, but on the central apparatus, which, however, the clumsy hypothesis of Dr. Brown-Sequard encourages us to overlook. But I cannot now tarry to give certain other anatomical and physiological reasons, for the belief that the view I have presented is the true one. Though it is not novel as I am well aware, yet it has not been utilized as it must and will be in the future.

Such are a few of the anatomical and physiological points to which I have felt it necessary to call attention, before passing to what are more commonly considered to be questions of pathology proper. It will be seen by what I have said, that in this case I lay very great stress on the central apparatus of sense, as compared with its so-called peripheral portion, or the nerve fibres. But what relations do such considerations sustain to neuralgia? It will be one aim in the remainder of my paper to try and give an answer to this question.

Neuralgia depends on a *material lesion*, however diffuse or slight, of some part of the sensory nervous apparatus. Of the correctness of this statement, I expect to offer proof as we pass along.

The form of general sensibility to which neuralgias in the proper sense of the word must be referred, *as a rule*, is the *sense of pain, or of painful impressions.* It is true that there may be changes in the sense of touch. It may be exalted or diminished. The former is properly called hyperæsthesia , and the latter anæsthesia in the present use of these terms. Then again, the sense of temperature may be increased or diminished. But there are no well recognized terms by which to express these variations in the sense of temperature. But it is hardly in reference to either of these forms of sensibility that the term neuralgia or *nerve pain* is used. It is true that there is a form of neuralgia in which the sense of temperature may be involved, and hence form a factor. I now refer more particularly to that form of burning neuralgic disorder, described by Dr. S. Weir Mitchell which he has denominated *causalgia.* But I do not now undertake to limit the meaning of the term neuralgia. There may be various forms of disorder of sensibility, both general and special, to be comprehended under the term. It is not my purpose to inquire into this subject now. It may be truly said, however that neuralgias as a whole, belong to the sense of pain, or of painful impressions. They occur mostly within this domain, and hence our attention 'will be directed chiefly to it. It may be either exalted or diminished. If the former we have strictly speaking *hyperalgesia*, not *hyperæsthesia*, if the latter, *analgesia*, not *anæsthesia.* It is true that the terms anæsthesia and hyperæsthesia might be so used in a generic sense, as to cover all cases of increased or diminished sensibility without violence to their etymological meaning. But they have been so loosely used and misused in medical writings, as to render a new nomenclature necessary for distinguishing different kinds of disorder of sensibility.

That these remarks are not arbitrary but founded on fact is apparent, when we remember that in many if not most cases of chronic neuralgia, the parts to which the affected nerve is distributed, have in a measure lost their sense of touch, as may be determined by the æsthesiometer. This is true, especially in many cases of trigeminal neuralgia. Often the slightest touch on the surface of the face within the region of distribution of the affected nerve, is the cause of excruciating pain. Nothing is more common than to speak of such cases as being hyperæsthesic, when in point of fact, a more careful investigation shows the existence of positive anæsthesia. There is however, in all such cases, *hyperalgesia* or a morbid increase of the sensibility to painful impressions, so much so, that impressions not at all painful in the normal condition of the sensory apparatus, produce in it pain. Now the question next in order is, what is the seat of the final lesion, whatever it may be, which must be one of the chief factors in a neuralgia? I have already tried to show that augmented sensibility cannot depend solely on any state of the related nerve fibres, but rather on the nerve centres, which are the real seats of sensibility, at least in its higher forms. Upon this point must we fix our attention; viz.: upon the condition of those groups of sensory cells, in which the sensory nerve fibres partly terminate, and which are fitted in a peculiar manner, at present unknown, to react to painful impressions. Now in what condition may we suppose these cells to be, when we consider the typical phenomena of neuralgia? It cannot be one of absolute loss or destruction of their proper structure. All the phenomena of the case forbid such a view. They must preserve their fundamental structure. The disorder of the cell groups to which reference is made, is not of an inflammatory nature. It is invariably, to come at once to the point, *a lesion of nutrition*, having its essential seat, so far as neuralgia is concerned, in those cell groups, which have the capacity of appreciating sense impressions of pain.

No other parts of the body are so bountifully supplied with blood for purposes of nutrition, as the nervous centres. This is because of their comparatively extraordinary functional activity. The lesion of nutrition referred to is simply one in which the cells have been worn from various causes, in their intimate structure, beyond the possibility of immediate repair by the regular process of nutrition, for this is the only way in which the repair can be effected. In some cases, for reasons that I will try to give hereafter, the lesion is practically irreparable. No organ can act, no cell can act, without a waste of its substance as a consequence. This process goes on ordinarily until it can go no further without endangering the integrity of the structure involved. At this point, that semipainful state sets in, which is called *fatigue*. If it is disregarded, and action is continued, the fatigue becomes painful, or even insupportable. This painful feeling is the natural inarticulate language, as we may say, of the worn tissue, and which may be regarded as a warning, that the process of waste or wear and tear, has been carried as far as it can be without endangering the fundamental integrity of the tissue. Ordinarily, when this point is reached, we obey the demand of nature, and *rest*. But rest simply means opportunity for repair of damage done.

When the repair is accomplished, we feel rested, as we say, the fatigue has given way to a sense of comfort, and the instincts and capacity for action begin to be manifested, to be followed sooner or later by a recurrence of that degree of intimate structural impairment, which gives note of its presence by the sense of fatigue. As far as the nervous system is concerned this nutritive lesion may not only take place in the motor tract, but the sensory as well. The action of the central motor apparatus or of the cells which gives rise to the motor impulse, which passes out along the motor nerves, no more certainly involves nutritional waste, than does the action excited in the cells of the sensory apparatuses by the sense impressions reaching them. The sense apparatuses become fatigued by continuous action or excitation, just as truly as the motor. If we rest them as soon as they become fatigued, they are repaired, and a sense of well-being comes as a result. But if for any reason the sensory tract, or any parts of the same, are over-excited by stimulation too intense, or too long continued, the process of nutritional impairment goes beyond the ordinary limit, and a painful or irritable condition sets in, as a testimony to overwear or excessive waste. The damage done is too profound to admit of repair in the time ordinarily allotted, or in any given case actually allotted for rest, or in other words, for nutritional repair. Hence the succeeding period of action or stimulation is begun, before the repair is complete. And this condition of things, if it goes on, as it often does go on, adds day by day to the preponderance of waste over repair, and hence to a permanent sense of fatigue, or in case of the sensory tract to irritability and pain, until a thorough loss of balance is sustained, which nothing short of prolonged rest and the best nutritional supply, and possibly the use of special means to quicken the processes of nutrition or, in other words, tonics, and the incidental aid of nerve sedatives, etc., can repair or restore.

In this worn and irritable state, it seems to me it is easy to see that any renewal of action or excitation will only increase the mischief and give rise to unpleasant symptoms, generally pain.

This process of gnawing, by nutritional waste, farther into the already worn and irritable nerve cells, as must happen, if action in them is renewed, provokes pain. This morbid process as it becomes, does not lead to the destruction of the apparatus, simply to its impairment as I have endeavored to describe. It is rendered irritable, hyper-sensitive to the impressions made on it or conveyed to it, by the nerve fibres,—it is unstable, too responsive, easily thrown into disturbance, difficult for it to suspend its activities when they are once aroused. But in whatever way it is produced, this is the state in which a nervous centre is, when it is in the "*neuralgic condition.*" I shall give ample though indirect proof of the correctness of this view hereafter.

There is no inflammation, no absolute dissolution of parts, simply the worn, irritable, hypersensitive condition described, not of the nerve fibres, (though they are often diseased in such cases) but of the sensory nerve cells of the central sensory tract, which is the sole seat of true nervous sensibility, as that term is ordinarily understood. This is *the one ever-present, essential factor, in all forms of neuralgia, whether general or local.* It is the background, the undertone in the picture, wherever seen.

Now in what ways may the condition of the sensory tract which we are contemplating, be produced? There are three principal modes of producing it, all of which may be present or not in any given case. The nutritive lesion about which I have spoken may be produced, first, by too much action or over-excitation, or second, by a deficient supply of nutritive materials, either with or without too much action, or third, it may be, as it often is hereditary, being recognized under the head of the "neuralgic temperament," or of "hereditary neuralgia." I will now consider these causal conditions in relation to the nutritive lesion which I have tried to describe as the fundamental element of neuralgia, irrespective of its form or locality. Then : 1. Those cases arising from peripheral over-excitation or irritation. These cases are divisible into two classes for practical purposes. *a.* Those in which there is no recognizable hereditary tendency to neuralgia, nor any marked deficiency as regards general nutrition, and in which the neuralgic condition of the sensory tract is produced chiefly by the severity and duration of the over-action or excitation, which sooner or later leads, in spite of the naturally healthy condition of the parts to that excessive tissue waste in which pain sets in, on comparatively slight provocation. *b.* Those cases in which the excitation or action may be very moderate, as measured by ordinary standards, but in which, either by reason of hereditary instability of the nervous structure, or by reason of a deficiency in the supply of nutritive materials or both conditions combined, as often happens, the same nutritive lesion; viz., an unhealthy preponderance of waste over repair is produced. For practical purposes only, these two classes deserve to be distinguished from each other, for at bottom they are the same. But, I wish now to fix attention for the moment, solely on the fact of peripheral irritation. If a sensory nerve fibre is healthy, every impression made on it is instantly transmitted to the centre to which it leads, and if the latter is in condition to receive the impulse, a more or less decided impression or disturbance is produced there. If the parts are healthy, and severe mechanical or other violence is done to the peripheral nerves, by which they are suddenly injured, the impression conveyed to the related centre, gives rise instantly to pain. But the pain is not necessarily to be explained in such a case by a reference to an abnormal condition of the centre, but on the simple ground of the peripheral lesion, and hence by the severity of the impression made on the centre which may have been at the time perfectly healthy. We attribute the pain solely to the visible external injury, as in cuts, lacerations, burns, etc. Such pain is not called neuralgic, though it is so in one sense, for it is nerve pain arising from nerve injury. But when very severe nerve pain *seems* to arise spontaneously, or is aroused by some slight touch or impression, never painful to a healthy organism, then it is called neuralgic. As I have said in substance, already, this condition of things cannot be explained except by going to the related nervous centre for the key of the explanation. But the condition itself in which the centre is found in such cases, cannot be understood except we take into account the action upon the centre of the sensory nerves which terminate in it. They are the natural channels through which the centres are excited to action.

If the impressions made on a given peripheral nerve, say the trigeminus, are severe, or, whether severe or not, are long continued and frequently repeated, the nervous centre becomes sooner or later in that state, as respects its nutrition, that I have already described, worn, sensitive, and ready to react with what we call pain, even to slight impressions reaching it by way of its nerves, and indeed in other ways as well, as I will endeavor to show hereafter. The requisite degree of irritation of a sensory nerve, may be produced in many ways, but generally by irritative disease of some kind. Generally the disease of the peripheral nerves arises, because they are involved in the structural diseases of the organs and tissues to which they are intimately distributed. Thus it happens to the trigeminus, in diseases of the teeth and jaws, which involve twigs or large branches of the trigeminus, and in diseases of the antrum, orbit, nasal fossae, in rheumatic, gouty, syphilitic, and other affections of the superficial and deep parts of the face, in injuries of the face and closely related parts, in severe exposures to cold, etc. In all these ways, and many others, may irritative disease be produced in the peripheral portions of the trigeminus, which more or less steadily transmits to the trigeminal sensory nucleus, that degree of irritating or exciting influence, which will, sooner or later, in different cases, cause that degree of nutritional disturbance, which I have called to your attention, as the fundamental factor in neuralgia. The nervous centres may be as easily worn, and with as bad results, if not worse, this way, as by over-action in a natural way. The same kind of action on particular sections of the sensory tract of the spinal cord, may be brought to pass, by disease of the mucous membrane of the stomach, or of the mucous membrance of any part of the alimentary canal, especially that of the lower part of the colon and rectum, and in various ways, as by dysentery, acute or chronic, especially the latter, by the habitual occupation of the colon, by hard and irritating fœcal masses, as happens in obstinate constipation, or in case of irritative disease of the kidneys, and more particularly of the bladder, as in chronic cystitis, prostatic disease, abnormal genital excitation, sexual excesses, irritative uterine and ovarian diseases of many kinds, disease of the liver, of the lungs, etc., etc. In all these cases, and many others, the visceral irritative disease almost necessarily affects the sensory nerves which ramify in the organs, and from these diseased nerves, a more or less steady tide of irritative and wearing, nervous impressions, is transmitted practically without cessation to certain parts of the sensory tract, to which the sensory nerves from any given part may go, and as a result, sooner or later, the central sensory apparatus of cells is brought into that worn, painful, irritable state, which I have described above. Every observing physician can recall from his own experience cases of this kind. We frequently find irritable and tender spines in the lumbar or lower dorsal regions, depending on irritative pelvic disease. Under such circumstances, the spine may be tender to the touch, with neuralgic pains radiating from the seat of irritation in the cord, downwards, or in other directions, differing in different cases? We also often find neuralgic pain come and go in association with gastric disease, either in the upper dorsal spine, or under one or both scapulæ, or around the course of certain of the intercostal nerves, or extending down the course of the brachial plexus, or its branches.

In all these cases the disturbed state of the nutrition of the sensory tract, or some definite part of it, is produced by excessive stimulation through the channel of the diseased nerves. The effects in the end are similar to those produced by overaction in a normal way. This state may be produced not alone by over-stimulation of the sensory nerves, or by irritative diseases affecting them, but by too long-continued, or too severe muscular exertion. Over-use of the arms in a variety of manual employments, over-use of the lower limbs, as in protracted standing or walking, or the use of the sewing machine, overstrain of the muscles of the back of the neck, as happens in the case of those whose occupations make it necessary for them habitually to bend the head forward, so as to put the muscles in question on a more or less continuous strain, etc., may bring about a similar condition, indirectly of the sensory tract. These protracted muscular efforts cannot be made without over-use of some part of the cord, and hence without involving the same nutritive lesion of the motor and sensory tracts combined, that I have already referred to. Nothing but rest, or in other words, repair will truly relieve such cases. Action must cease that waste may cease, and that repair may go on without disturbance.

But the nutritional lesion, about which I am speaking, may be produced in other ways, as well as those already described.

One of the most prolific sources of the neuralgic condition, is defective nutritive supply. But few conditions are more common than this. How often do we find cases, in which by reason of privation of food, or disease of the digestive system, which impairs the appetite, or if not so, impairs digestion and hence disturbs the digestive process and vitiates its results, or where by choice or habit, unsuitable or unnutritious food is taken, and hence, in one way or another from the side of supply, anæmia is produced? Then, again, how often do we find cases in which though digestion is good, and the supply of food fair, yet by reason of some excessive discharge as by hemorrhage, impoverishment of the blood occurs leading to that worn, irritable condition of the nervous system as regards its nutrition, which is the prolific soil out of which the numberless forms of neuralgia may spring. Under such circumstances a local neuralgia is often produced by very slight, local, irritative action, or over-action, for the essential background is in such a case gratuitously supplied. It takes but a comparatively short period in time to produce that irritable hyper-sensitive condition in the sensory tract, which is found at the bottom of every neuralgia proper. And when the neuralgia is once under way, in such a case, there is but little prospect for a recovery, while the plain conditions on which it depends are unrecognized or remain the same. In every case of neuralgia, the condition of nutrition of the patient should be made the subject of the sharpest inquisition.

One of the commonest forms of unrest, and hence of excessive waste, takes place from loss of sleep. This is at bottom not anywise different trom the form of nerve wear and tear, already described. It simply deserves a mention for practical purposes. Sleep is brain rest, or it is nothing. If so, then lack of sleep entails, of course, that preponderance of waste over repair, and hence the neuralgic state with which we are already familiar. In this way do many if not most painful or neuralgic conditions of the brain partly arise. The pain growing out of this impaired nutritional state of the brain, may be general or local, slight or severe, paroxysmal or continuous, as I have often seen. It is not even necesary that we should have pain physiologically speaking. It may be what has been called "psychical pain." It may be a painful emotional state, such as we see in distressing forms of melancholia, a kind of pain, if it can be called so, sometimes harder to bear than mere physical pain. I am much inclined to agree with Krafft-Ebing, who calls melancholia a "psychical neuralgia." In my opinion it arises out of the same condition exactly as regards brain nutrition,—say of the cerebral cortex,—as that which I have described as the fundamental organic basis of neuralgia. Essentially the same causes produce both, and they are relieved by similar means for cure. But I cannot speak on this interesting topic to the extent it deserves. But one of the chief causes of the impaired nutrition of brain, on which the neuralgias, more particularly of the brain depend, is the loss of sleep. I know very well this is not new, and I also know it is seldom practically heeded and thought about, as it certainly deserves to be.

Then let me once again, recall to your notice, that the unstable condition of nervous system, as regards its nutrition, its solidity and perfection of structure, may be hereditary, and if so, is a serious, even an irremediable matter. It is necessary above all else, to recognize this factor, in the respect to prognosis. It will save you much trouble and confusion, for it will often prevent you from making promises, or at least from exciting hopes for recovery, destined, as might be seen before hand, not to be fulfilled. It will be of value also, to recognize the hereditary factor in pointing out specific hygienic courses to our patients. They cannot without risk, do or endure, what others may with impunity.

But one of the more prolific immediate causes of neuralgic attacks, is fluctuation in blood supply. There are two ways in which these fluctuations may operate. If there is too little or greatly too much blood supplied to a nervous centre, in a given time, there must be some change in its nutrition, generally a diminution to a point below the normal rate, and if so, a tendency toward that condition of nutritional impairment, which has been described as the most essential pathological factor in neuralgia.

Not to speak at present of congestions, and the nutritive disorders to which they may be supposed to lead, I would call your attention for a few moments to the opposite condition—anæmia. It has been supposed by some writers, that we may and do have a condition of the circulation of even quite limited tracts of the cord or brain, in which through excitation of the related vaso-motor nerves, the vessels are made to contract to much less than their normal size, and hence, in this way, to exclude a portion of the blood, which they should normally convey to the part to which they lead. It is held by those, who are of this belief, that this condition of vasal spasm may endure for even months in extreme cases, without any considerable periods of relaxation. Now if this condition does actually occur, and I believe it is possible, it can be seen by you all, how it might naturally lead to impaired nerve nutrition, and hence to the neuralgic condition. This condition of the circulation, it is maintained by my friend, Dr. Hammond of New York, occurs in the so-called spinal irritation, to which I have referred incidentally, already. He supposes that in spinal irritation, there is anæmia of the vessels of the *posterior columns* of the spinal cord, produced by vasal spasm. If this actually happens, it would lead to the neuralgic condition I have described. It is not my purpose this evening to discuss the view of Dr. Hammond, to which I have alluded, but I will remark in passing, that while I think the circulatory anomaly *possible*, yet I doubt its existence in such cases. There is no real proof of the correctness of the hypothesis. It is simply inferred from a group of phenomena, which may be explained in a better way, so it seems to me; viz., by reference to a nutritional lesion, to explain which, such a hypothesis is not necessary. But admitting that such circulatory disorders may and do occur, it is easy to see how they might conduce to bring about the "neuralgic condition." But it is not so much to changes *in the rate of nutrition*, brought about by quantitative changes in blood supply, to which I wish to direct your attention this evening, but rather to *changes in blood pressure within the cranio-spinal cavity and the results of the same.*

I know very well what are the opinions which have been entertained in relation to the question, as to whether the quantity of blood within the cranio-spinal cavity is, or may be subject to much variation. I know it has been held by some, that being air-tight unyielding cavities, into which the blood is forced by atmospheric pressure, on principles well understood, in other relations, there could not be any appreciable variation in quantity of blood at one time, as compared with another. If you have more blood in any given part of the brain at one time, it must be for the reason that some other part or parts have in proportion less than they should have. This is one position, and it is true, as I believe, to a degree, which does not obtain for other organs not situated similarly. I know also that the contrary opinion has been and is now maintained. It is also partly true, according to my view. But for my present purpose, this question may be put out of sight altogether.

If it is doubtful whether there can be more blood in the brain, as a whole, at one time than at another, there can be no doubt, so it seems to me, as to whether there may be more *pressure*—vascular pressure—at one time, than at another in the cranio-spinal cavity. It may, and in point of fact, does vary, greatly. There can be no question in my own mind after a pretty careful study of the subject, that the situation of the brain in an airtight unyielding cavity, insures a steadiness and fullness of blood-supply, not possible under different physical conditions. This is necessary in the case of organs, of such high vascularity, and having such delicate and important functions. But for various reasons, not only the quantity, but the pressure of the blood in or into the brain and cord, may vary. Vascular pressure within the cranio-spinal cavity, may be changed, either in one or the other of the following,—among other ways :

1. By changes in vascular tonus, affecting the energy of the heart's action. The action of the brain and cord may be deeply affected by a loss of energy of the heart, by which vas-

cular tension within the cranial cavity is diminished, giving at times,—that is if the brain is in that condition as respects its nutrition, which I have called neuralgic,—those severe neuralgic headaches of anæmia, which are aggravated by the upright, and are made better by the recumbent, posture, and by the use of certain remedies, amongst them such as will excite the heart to stronger action, and will hence increase vascular tension, especially within the cord andbrain. In such cases, alcoholics, quinia, the preparations of opium, &c., are found beneficial. But if the brain should be in the worn, neuralgic condition I have supposed, any condition which would increase the force of the heart's action, and hence suddenly increase vascular tension, will often cause pain in the head. This is seen in the effect of strain in severe physical effort, which increases venous tension, and also in any rapid exercise, which temporarily increases the force and frequency of cardiac action, and in the posture of stooping in such way as to cause blood to flow more freely to the head, so as to increase blood pressure, and finally in the throbbing character of such pains, the throbs corresponding to the heart beats, and further in the relief produced by the means calculated to diminish the force of cardiac action, or in any other way by diminishing vascular tension, or contributing to a quiet and steady circulation.

Then, again, vascular tension within the cranio-spinal cavity may be produced by means of certain climatic changes, more particularly the following: In the first place, by changes in temperature. Either extremes of heat or cold, if they are suddenly brought to pass, may change blood tension in the cranio-spinal cavity. If the surface is suddenly exposed to cold, the skin and its small vessels contract, and as a consequence, less blood circulates in the surface, than is necessary to preserve an equilibrium, as between superficial and deep-lying parts, and hence more blood circulates in the visceral cavities, or tends to do so, than under different circumstances. Or, on the contrary, if the surface is more or less suddenly exposed to a high temperature, the cutaneous vessels dilate and admit more blood to the exterior of the body, and hence less relatively goes to the interior, thus giving rise frequently, though not necessarily, to diminished vascular tension, in the cranio-spinal cavity. Under such circumstances, it is very common for a person to complain of temporary loss of nerve power, or languor, and other symptoms which can hardly be ascribed to mere changes in nutrition, but in part, at least, to changes in pressure. Under such circumstances, neuralgias, if the neuralgic condition exists, are very likely to set in on slight provocation.

In the second place, changes in barometic pressure, give rise, under the peculiar circumstances of the circulation in the cranio-spinal cavity, to changes in vascular pressure. This may be seen in extreme forms in the effects of high altitudes on the nervous system, and in the effects of increased atmospheric pressure, a good example of which is seen in the case of workmen operating in the condensed air of the caissons beneath the piers of certain bridges. The atmospheric pressure on the surface varies greatly from time to time, as is well known, and certainly leads to changes in vascular pressure within the air-tight, unyielding cavity, in which brain and cord lie. This, as I believe, from pretty close observation, is *one* of the prime exciting causes of the nervous symptoms,—and neuralgia is chief among them,—which seem to depend on the "weather." This view will also aid in explaining why steady, mild climates, are, as a rule, the most favorable to the removal of those nervous symptoms, which appear to be influenced by climatic changes. Within the past year, I have seen some unpublished charts, drawn up by Dr. S. Weir Mitchell, of Philadelphia, in which he had made the endeavor to trace, by means of lines which resembled isothermal and magnetic curves, the relations of neuralgia to certain climatic changes. I do not know what conclusions he may have reached, but it is a subject to which I have given considerable attention, and I am convinced that much of the influence exerted by climatic changes on certain nervous affections depends on variations in vascular pressure, produced by temperature and barometric changes operating in the way I have just described. I am far from thinking that these or similar exciting causes, can produce neuralgias in a healthy nervous system, but that they may do so, in cases where the "neuralgic condition" already exists, as it so often does.

But without exhausting these practical considerations as to the causes or conditions of neuralgia and their modes of operation, I will call your attention finally to what I consider a most important means for producing changes in vascular pressure in parts of the sensory tract. I now refer to *disordered vaso-motor action.* The muscular vessels, notably the small arteries, just previous to their termination in the capillaries, are abundantly supplied by vaso-motor nerves, which are distributed to the muscular tissue in their walls. This muscular tissue is under the control of the vaso-motor nerves in question, just as truly as the voluntary muscles are under the control of the ordinary motor nerves distributed to them. The vaso-motor nerves in a certain sense—but in a certain sense only—arise from vaso-motor centers, situate somewhere in the gray matter of the spinal cord and medulla oblongata. These centers are taken as a whole in close relations with the sensory tract of the cord and medulla, and what may be called the "emotive" tract of the cortex of the cerebrum. Excitation of the sensory or "emotive" tracts, in a reflex way, excite the vaso-motor tract in the cord and medulla, just as truly as the motor tract proper may be, from which the motor roots of the spinal nerves proceed. In this way; viz., by reflex action, any part of the vaso-motor tract in the cord and medulla may be excited by means of influences reaching it by way of the sensory tract proper of the cord and medulla, or from the cortex, as a result of emotional or other excitement. Excitation of the central vaso-motor tract, or any part of it, may and does give rise to an outgoing motor impulse, along the related vaso-motor nerves leading to some definite locality, some definite vascular area, which may be exceedingly small, or on the contrary, very large. This vaso-motor impulse aroused in a reflex way, passes as just said, along the vaso-motor nerves which are distributed to a certain vessel, or its branches, and suddenly they dilate or contract as the case may be, and in this way is blood-pressure suddenly, even violently, changed, in some particular locality—external or internal—of the body. Almost innumerable examples can be given. A familiar instance of this kind of action was shown a long time ago, by Brown-Sequard and Tholozan. It consisted in dipping one hand in ice cold water, and maintaining it there for a time, while the other grasped a thermometer. In a short time a very considerable fall in temperature of the hand not in the iced-water, was indicated. By repeated test experiments, it was proved that the fall in temperature of the one hand was due to the fact of the severe chilling of the other.

The explanation can be readily given in view of what has been said as to the mechanism and mode of action of the vaso-motor system. The severe impression made on the sensory nerves of the hand in the cold water, was transmitted to the upper dorsal and lower cervical cord, and transferred to that portion of the central vaso-motor tract situated there, and in a reflex way an impulse was sent along down the vaso-motor nerves of the other arm, which caused sudden contraction of the muscular vessels, to which these nerves are distributed, and as a consequence, the supply of blood to that arm was suddenly diminished, this caused, very naturally a partial cessation in nutritive activity, and hence finally a diminution in the amount of heat produced, the production of which depends on tissue change, and this is the explanation of the fall of temperature in the arm. But the point to which I wish to direct your attention, is not the fall of temperature but rather to this mode of suddenly changing the vascularity, and hence the blood-pressure of a part, more especially the sensory tract of the central nervous system.

There is one point in connection with this subject to which I desire to call your especial attention. It is that changes in vascularity in the brain and cord produced by the channel of the vaso-motor nerves, may be, and frequently are in the strictest sense of the word local. Very small portions or regions of the brain or cord, may, by the means now under consideration, become the seat of an intense congestion without surrounding parts being to a similar degree, or at all affected. These "regional" fluctuation in blood supply are of very great importance in the physiology and pathology of the nervous system.

I have good reasons for thinking they are one of the chief factors, especially in typical or periodical, and occasional neuralgias, such as are due to malarial influence, in chronic hemicrania, and other particular forms. In hemicrania the attacks occur once a week, or once a month, or at other intervals. I am fully persuaded in the first place that the sensory nucleus of the trigeminus, in part or altogether, on one or both sides, is the seat of that form of nutritive lesion which I have described as the most essential factor in a neuralgia, and that at the time of the attack it becomes the seat of a sudden local change in vascularity, and hence of blood-pressure, which produces that degree of physical shock in the painful irritable center, which is the immediate cause of the pain. This may and does occur often, without the slightest sign of peripheral disturbance as for example in hereditary cases. The attacks often come on, and pass off, too suddenly, to enable us to explain them in any other way, so it seems to me, than by a reference to a local disturbance of the circulation. In making the statement that attacks of hemicrania often come on which cannot be referred to any adequate peripheral disturbance, I do not forget the opinions which have been emitted by writers like Du Bois Reymond, Moellendorf and others. The former in his studies of hemicrania as it occurred, unhappily in his own person, found that the region to which the pain was limited, (the first division of the fifth pair on one side) was pale and cold as compared with the opposite side, and the temporal artery which supplied that region, was not only smaller but harder under the finger than natural.

His opinion was, that the pain was caused by spasm, of the branches of the temporal artery, produced by irritation of its vaso-motor nerves. The firm contraction of the muscular tissue of the vessel, it was supposed, compressed or pinched the sensory filaments of the trigeminus, and so produced the pain. Du Bois Reymond seems to have been almost solely occupied with a consideration of the condition of the *peripheral* termination of the nerve supplying the affected region, rather than with the condition of the center, which, in my opinion, is in all such cases the chief seat of disease. As proofs that his view was incorrect, it may be remarked that soon after, Moellendorf described a form of hemicrania in which the opposite condition of the vessels in the painful region is found, and also that cases occur in which there is no appreciable external vasal disturbance, and that it often happens that quite as marked vaso-motor disturbances occur in the same region without producing pain at all. These and other reasons seem to disprove the correctness of that view, which ascribes the pain to peripheral vaso-motor disturbance. No doubt when the center back to which the nerves from the affected region lead, is in the painful condition I have described, earlier in this paper, any peripheral disturbance, however slight, may excite pain. But this should not mislead us, as it has so often done, so as to attract our attention from the center, which is from first to last the most essential seat of disease. This being so, we are not left in a state of surprise at the continuance of a neuralgia, after the removal of the peripheral irritation, in which it in part had its beginning, even after section of the nerves which are the apparent seat of the neuralgia. The reason why such means are sometimes not effectual, is because the real seat of the essential disease is not where it is commonly supposed to be; viz., *in the peripheral nerves* of the affected region, *but in the center.*

One means among others for affecting painfully these diseased parts of the sensory tract, no matter where they are, is sudden and violent changes in blood pressure, and one important way for producing this state is by disordered local vaso-motor action, brought about in a reflex way. The starting point of the irritative action, may be in any part of the peripheral nervous system, at the skin or mucous membrane of the stomach, or of other sections of the alimentary canal, the uterus or ovaries, or, on the other hand, from the cortex of the brain, by means of emotional excitement, for the cortex is, in my judgment, the seat of the emotions proper. But from either or both these directions, and in a multitude of ways, may the exciting or irritative agencies act which may give rise to the impressions which, when they are conveyed into the sensory tract, may be transferred to closely related vaso-motor centers, from which, in turn an impulse may be emitted along the out-going vaso-motor nerves, which produces in the way already described the changes in vascular pressure, which, probably, in a mechanical way, gives rise to the central pain, which is referred to the periphery of the related nerves in accordance with the "law of eccentric projection" of Romberg.

But here I must terminate my summary of conditions and causes of neuralgia, without completing it. I have said enough to indicate somewhat clearly, I hope, my views as to the pathology of neuralgia.

The subject is becoming more important every year, among civilized peoples, for the tendency seems to be, that painful affections of the nervous system increase as civilization becomes more refined and artificial, and the occupations of the race more intellectual and emotional, and the domain of sensual enjoyment and excess, more extended.

From the foregoing discussion, we may formulate perhaps usefully the following general results, or conclusions:

1. Neuralgia has its essential seat in the central apparatus of sensory cells, called the sensory tract, lying within the confines of the gray matter of the cerebro-spinal axis. Its essential seat is not in the peripheral nerves. Hence, it is incorrect and misleading, to speak of, or describe, neuralgias as "diseases of the peripheral nervous system."

2. The essential morbid condition in a neuralgia, is a nutritive lesion ot the central sensory apparatus of cells, which are the seats of true nervous sensibility. This state is frequently caused by disease of the peripheral nerves, but even in such cases the more irritable state of the sensory tract is the main factor reached by judicious analysis of the phenomena of neuralgia. In this condition it reacts with pain to even trivial impressions made on the sensory nerves, which terminate in the affected region.

3. The attack of pain may be due to overexcitation, and hence over-wear and waste of the affected center, produced by the channel of its sensory nerves, or by changes in blood-pressure in the affected center, caused by loss or increase of tonus of the peripheral vessels, or a change in cardiac action, or by changes of posture, or of temperature, or of barometrical pressure, or by influences acting on the vaso-motor nerves, distributed to the diseased center, and which may be affected from either the peripheral nervous system, or from the cortex cerebri.

Many proofs could be given of the substantial correctness ot the views I have set forth, besides those already offered, but I do not have time to give them in this paper, already beyond ordinary length.

Passing by the proofs referred to, I wish to call your attention for a few moments, in conclusion, to the treatment of neuralgia. It is one of the highest tests of the truth of a pathological doctrine, when it points, as it were, *a priori*, clearly to the true line of remedial action to be adopted. Judged by this standard, the pathology of neuralgia, as I have endeavored to expound it, must take a higher rank than that of a mere hypothesis, to account for the phenomena of the case. If the above described doctrine, as regards the intimate nature of neuralgia, is true, it points naturally, and inevitably, to three prime indications for treatment. They are as follows:

1. *Rest, or cessation of action or excitation.* This is plainly required by the supposed worn, or wasted, irritable condition of the sensory tract, or some limited part of it. It is one of the clear dictates of the case. In point of fact, nothing is more important in the treatment of neuralgia than the fulfillment of this indication. It is not appreciated as it should be in the profession. The necessary quiet of the diseased center may be secured, in many ways, according to the source and kind of irritation or excitation. It prescribes that all irritating substances or diseases shall be removed at once whenever possible, even if there must be division of the sensory nerve trunks, of the affected region, to cut off from the diseased center, all peripheral excitation. It prescribes that all central disturbance, such as arises from changes of blood pressure and other causes in the diseased sensory tract, shall be remedied.

2. It prescribes that inasmuch as there is too much waste, in the sensory apparatus, as the prime condition of the neuralgia, that every means should be employed; (*a*) to supply the greatest amount of nutritive material of the best kind for the purpose of repairing the supposed nutritive lesion; (*b*) that everything which may be faulty in the action of the organs, or in the processes on which nutrition depends, must be corrected, especially by the use of alteratives, and of

tonics which are best calculated to facilitate or hasten the nutritive processes, and, in this indirect way, to increase strength.

3. Lastly, it prescribes that some means shall be employed as occasion arises, to allay the abnormal nervous excitability, or the pain, for the sake of immediate relief, or to give that tranquility, so much needed, and which it is often impossible to obtain, except by artificial means.

Now, these are the prime indications clearly dictated by the pathological conditions exposed, and it so happens that they are exactly those gradually sanctioned by ages of the best practical experience in dealing with neuralgia, prior to any correct notions as to its pathology. But with this outline of views, I must close my somewhat hasty paper. You will observe that no claim of novelty is made in behalf of the views presented. They are presented simply because they are suggestive, and believed to have, if thought on, greater practical value than they have been supposed to possess.

ART. II.—PATHOLOGY AND TREATMENT OF REFLEX MOTOR SYMPTOMS—PARALYSIS, CONTRACTIONS, ETC.

A DISCOURSE BEFORE THE COUNTY MEDICAL SOCIETY OF NEW YORK, Jan. 8th, 1877.

BY EUGENE DUPUY, M. D.
(Member of the American Neurological Association; Corresponding Member, Paris Societe de Biologie, etc., etc.)

Paralyses from destruction of conductors only take place in the spinal cord. Yet even when conductors are diseased or destroyed in that organ, the palsy which is observed is mostly not due to the loss of function of the parts destroyed, but to an influence exercised by the diseased portion upon other areas of the cord. Provided a very few fibres are left healthy, the